Thiamine Deficiency in a Team of Sled Dogs

Doreen M. Houston and Thomas J. Hulland

Thiamine deficiency has been reported in a wide variety of species, including cattle, horses, dogs, cats, foxes, mink, and man. Animals with a high requirement for thiamine, such as cats, mink, and foxes, are commonly affected. Such animals are often fed species of raw fish which contain thiaminase (1–3).

In the dog, reports of naturally occurring cases of thiamine deficiency are scarce.

Most commercial dog foods provide more than the daily 22 μg thiamine/kg required by an adult dog (4). Meat, especially pork and liver, and cereals such as oatmeal, whole wheat and yeast are good sources of thiamine (4–6). Thiamine is also obtained from bacterial synthesis in the gastrointestinal tract (4). Small amounts of excess thiamine are stored in the heart, kidney, and skeletal muscle (7). Puppies, working dogs, pregnant bitches, and geriatric dogs require increased amounts of thiamine in the diet (4).

Thiamine is heat-labile and water soluble and may be destroyed by cooking. Over-processing of commercial dog foods, the feeding of raw frozen fish, and the ingestion of thiamine antagonists have been implicated in causing thiamine deficiency in the dog (8–12). Deficiency syndromes generally develop weeks after the initiation of a thiamine-deficient diet. Experimentally-induced thiamine deficiency in Beagle dogs has been studied and clinical and necropsy findings were similar to those of the clinical cases examined in the present study (13,14).

In the fall of 1984, on a small farm in Southern Ontario, 12 sled dogs began to lose weight. For six months prior to the onset of illness, the dogs had been maintained on water and fresh frozen cleaned carp and suckers caught locally in a small commercial fishing operation. No supplementation had been given. In previous years, particularly during the winter months, fresh horse organs were also provided. This was not done in the current year. The dogs were tethered outside all year round. They had been vaccinated against rabies three weeks earlier and dewormed for tapeworms (Canoids — MTC Pharmaceuticals, Canada Packers Chemicals, Mississauga, Ontario) a few days prior to the onset of clinical signs.

One dog, a two-and-one-half year old intact male Collie cross had been totally anorectic and lethargic for two days. Paraparesis began on the third day and progressed to spastic tetraparesis over the next six hours. Trismus was present. Within 24 hours of the onset of neurological signs, the dog began to convulse, became comatose, and died. The animal was presented to the Ontario Veterinary College for necropsy.

Department of Clinical Studies (Houston) and Department of Pathology (Hulland), Ontario Veterinary College, University of Guelph, Guelph, Ontario N1G 2W1

An all-fish diet high in thiaminase depleted the reserves of thiamine and resulted in the clinical signs

A second dog, a one-year-old intact male Collie cross followed a similar course to the first. Anorexia was apparent for two to three days, during which time the dog appeared to be constipated. He stood with pelvic limbs abducted and the tail rigidly flexed. Following this period, the animal developed progressive spastic tetraparesis and in this state was referred to the Ontario Veterinary College. This dog was thin, had opisthotonus and was hyperesthetic in the hindquarters. Temperature, heart rate, and respiratory rate were 38.0°C, 120 beats per minute and 64 breaths per minute respectively. The pupils were widely dilated and both the pupillary light response and menace reflex were slow. Within minutes of being handled, the dog went into tonic, clonic convulsions. Differential diagnoses included exposure to toxins (lead, mercury, cyanide, or carbon monoxide), viral encephalitis (distemper, rabies, or post-vaccination reaction to rabies) and metabolic derangement (thiamine deficiency).

Prior to euthanasia, samples were obtained for a complete blood cell count (CBC), biochemical profile, urinalysis, and blood for lead determination. A sample of cerebrospinal fluid (CSF) was obtained at the time of death.

The CBC results revealed a hematocrit of 54.7 L/L (normal 37–55 L/L) and total serum protein of 69.3 g/L (normal 52–69 g/L). Abnormalities detected on the metabolic profile included urea 25.0 mmol/L (normal 1.8–10.4) and creatinine 111 μmol/L (normal 60–110 μmol/L). Urine specific gravity was 1.068. These results were compatible with prerenal azotemia. Mild muscle damage was reflected by a creatine kinase value of 468 U/L (normal 30–250) and aspartate aminotransferase of 363 U/L (normal 8–38). Blood levels for lead were normal.

The sample of cerebrospinal fluid had a nucleated cell count of 0.003 × 10^9/L (normal 0–0.005) with

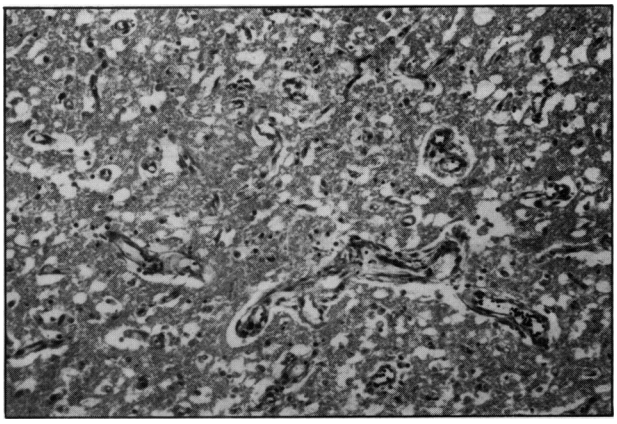

Figure 1(a). Marked prominence of blood vessels and neovascularity in the center of an area of malacia. H & E.

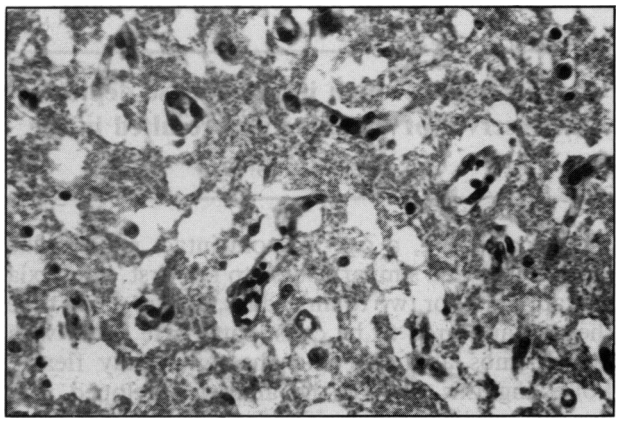

Figure 1(b). In an area of greatest destruction, myelin sheaths and axons have disintegrated leaving open spaces and debris. H & E.

mononuclear cells predominating. There was 0.65 g/L of protein present (normal less than 0.22 g/L). The interpretation made was chronic nonseptic degenerative encephalitis. Brain submitted for rabies analysis was negative on fluorescent antibody examination (Health of Animals, Nepean, Ontario).

Gross lesions were not visible in the dogs presented for necropsy. Histological lesions in brain were confined mainly to the rostral and caudal colliculi of the corpora quadrigemina. The caudal colliculi were the most severely affected structures in both dogs (Figures 1 (a) and (b)). Well demarcated areas of malacia as well as an intense gliosis and neovascularity were present bilaterally. Arteriolar walls in the area were markedly hypercellular and were accompanied by a few gitter cells. The small myelinated fibers had the most damage with a few neurons also showing degenerative change. The rostral colliculi, lateral lemniscus, and rostral cerebellar nuclei had less severe involvement with edema, astrogliosis, scattered degenerated fibers, and hyalinization of small arterioles evident. A diagnosis of chronic thiamine deficiency was made.

The remaining ten dogs on the farm were exhibiting decreased appetites, dullness, and a few had a broad-base stance.

The owners were advised to feed cooked fish and to introduce oatmeal and dry dog food to the feeding regime. As well, the dogs were given 100 mg thiamine intramuscularly and were fed 100 mg thiamine daily for two months. No evidence of thiamine deficiency was seen in these dogs during the next two years.

Thiamine is a B-complex, water soluble vitamin. It plays a fundamental role in the intermediate carbohydrate metabolism of all living cells (4). The primary metabolic consequence of thiamine deficiency is a widespread reduction in cerebral glucose utilization (15). Because the central nervous system (CNS) depends on the metabolism of carbohydrates for its energy supply, thiamine deficiency can greatly impair the function of the CNS.

In carnivores, the histological lesions of thiamine deficiency are usually confined to the brain stem periventricular grey matter, which includes the lateral geniculate nuclei, oculomotor nuclei, caudal colliculi, and vestibular nuclei (15,16,17). This differs from other species in which cortical involvement is more pronounced. In the dog with thiamine deficiency, paresis and paralysis are common.

In the dogs discussed in this report, an all-fish diet high in thiaminase depleted the reserves of thiamine and resulted in the clinical signs. Thiaminase is known to be high in many species of fish including carp and sucker (12,18).

The greatest antithiamine activity in fish is found in viscera, particularly spleen, liver, intestines and heart (5,6). The fish here had been eviscerated prior to freezing and this may explain why the dogs' thiamine reserves were not depleted much earlier. If the owner had continued supplementing the diet with fresh horse organ parts, as he had in the past, clinical signs of thiamine deficiency may not have occurred.

References

1. Loew FM, Martin CL, Dunlop RH, Mapletoft RJ, Smith. Naturally occurring and experimental thiamine deficiency in cats receiving commercial cat food. Can Vet J 1970; 11: 109-113.
2. Jarrett J. Thiaminase-induced encephalopathy. Vet Med Small Animal Clin 1970; 65: 705-708.
3. Loew FM, Austin RJ. Thiamine status of foxes. Can Vet J 1975; 16: 50-52.
4. Mellentin RW (Manager, Technical Services). Gaines: Basic Guide to Canine Nutrition. 4th ed. New York: Gaines Professional Services, 1977.
5. Phillips RW. Water-soluble vitamins. In: Jones LM, Booth NH, McDonald LE, ed. Veterinary Pharmacology and Therapeutics. Ames: The Iowa State University Press, 1977: 779-786.
6. Abrams JT. Animal Nutrition and Veterinary Dietetics. 3rd ed. Edinburgh: W. Green and Son Ltd., 1961.
7. Noel PRB, Chesterman H, Jolly DW. Thiamine (Vitamin B_1) Supplementation in the dog. Vet Rec 1971; 89: 260-266.
8. Read DH, Jolly RD, Alley MR. Polioencephalomalacia of dogs with thiamine deficiency. Vet Pathol 1977; 14: 103-112.
9. Mahyew IG, Stewart JM. Nervous syndrome in dogs possibly associated with thiamine deficiency. Vet J. 1969; 17: 91-92.
10. Harapanhalli GN. A case of lumbar paralysis in dog. Indian Vet J 1975; 52: 491-493.
11. Ahrens FA. Thiamine deficiency in puppies. Mod Vet Pract 1980; 61: 568.
12. Putney WW. Chastek paralysis in dogs. J Am Vet Med Assoc 1945; 106: 164-165.
13. Read DH, Harrington DD. Experimentally induced thiamine deficiency in beagle dogs: Clinical observations. Am J Vet Res 1981; 42: 984-991.

14. Read DH, Harrington DD. Experimentally induced thiamine deficiency in beagle dogs: Clinicopathologic findings. Am J Vet Res 1982; 43: 1258-1267.
15. Hakim AM, Pappius HM. The effect of thiamine deficiency on local cerebral glucose utilization. Ann Neurol 1981; 9: 334-339.
16. Oliver JE, Lorenz MD. Handbook of Veterinary Neurologic Diagnosis. Philadelphia: WB Saunders Co., 1983.
17. Delahunta A. Veterinary Neuroanatomy and Clinical Neurology. 2nd ed. Philadelphia: WB Saunders Co., 1983.
18. Hirn J, Pekkanen TJ. Quantitative analysis of thiaminase activity in certain fish species. Nord Vet Med 1975; 27: 646-648.

Prevalence of *Nematodirus battus* in Sheep in New Brunswick and Nova Scotia

Harry J. Smith and Shiona McIntosh

In 1986, *Nematodirus battus* was recovered from sheep for the first time in Canada on two farms in Prince Edward Island (1,2). From September to December, 1986, a study was carried out on lambs going to slaughter from Nova Scotia and New Brunswick to determine the prevalence of this worm in sheep in the Maritimes. Lambs originating from 35 farms in eight counties in Nova Scotia and from seven farms in three counties in New Brunswick were examined. A rectal fecal sample and the first 6 m of the small intestine (including the duodenum) were collected from each lamb. Intestines were submitted either refrigerated or fixed in 10% formalin depending upon the method of transportation used.

Fecal samples were examined for the presence of *N. battus* eggs by the simple flotation procedure and a saturated nitrate flotation solution. Worms were recovered from the small intestine by slitting the gut with scissors, washing contents and mucosa vigorously in water, and passing the washings through two sieves (mesh sizes 20 and 35). Eggs and worms were identified on the basis of morphological characteristics described previously (2,3,4).

On the average about twice as many lambs were examined from each New Brunswick flock compared to each Nova Scotia flock. From 12 flocks in Nova Scotia and two flocks in New Brunswick, only single lambs were available. Fifty-seven (63%) of the 90 lambs from Nova Scotia and two (7%) of 27 lambs examined from New Brunswick were infected with *N. battus*. Infected lambs were found in 21 flocks originating in Antigonish, Colchester, Cumberland, Hants, Pictou, and Victoria Counties in Nova Scotia, and in two flocks in Westmorland County in New Brunswick. Of the 59 infected lambs, eight (13.6%) were diagnosed by fecal examination, 24 (40.7%) by recovery of worms, and 27 (45.8%) by both methods.

Animal Pathology Division, Health of Animals Directorate, Food Production and Inspection Branch, Agriculture Canada, P.O. Box 1410, Sackville, New Brunswick E0A 3C0 (Smith) and Department of Biology, Mount Allison University, Sackville, New Brunswick E0A 3C0 (McIntosh).

It is evident that *N. battus* is prevalent in sheep flocks in the Maritimes

Thus about 86% of the positive cases were diagnosed by recovering the worms while about 59% were diagnosed by fecal analysis alone. It is likely that additional positive findings would have been revealed if the lambs had been examined earlier in the season when they were younger and presumably had larger worm burdens (5).

In summary, it is evident that *N. battus* is prevalent in the Maritimes, existing in sheep flocks from Victoria County in Cape Breton Island to Westmorland County in New Brunswick. The clinical significance of *N. battus* under Canadian conditions is not known.

References

1. Smith HJ, Hines JG. *Nematodirus battus* in sheep. Can Vet J 1987; 28: 18.
2. Smith HJ, Hines JG. *Nematodirus battus* in Canadian sheep. Can Vet J 1987; 28: 256.
3. Crofton HD, Thomas RJ. A new species of *Nematodirus* in sheep. Nature 1951; 168: 599.
4. Hoberg EP, Zimmerman GL, Pickard LG. *Nematodirus battus*: A review of recent studies relative to the development of surveillance and control programs. Proc US Anim Health Assoc 1985; 89: 420-431.
5. Dunn Am. Veterinary Helminthology. Philadelphia: Lea Febiger, 1969.